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CASE REPORT

Crush syndrome presenting three days after injury

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Introduction

Crush syndrome is due to extensive muscle damage characterized by haemodynamic shock, hyperkalaemia, metabolic acidosis, and myoglobinuric acute renal failure. The literature is somewhat divided on its treatment; some surgeons opting for late surgery while others maintain that early surgical intervention is best. We report a case of a young man with a crush syndrome presenting 3 days after his leg was caught in a train door and examine the current literature on the topic.

Case presentation

A 22-year-old Latin American man attempting to switch trains near the Texas–Mexico border accidentally got his right leg caught in the train's sliding doors when they closed. He remained trapped outside the train for approximately 3 days prior to presentation at a regional Level I Trauma center. He had had an unknown surgery to his left hand, but otherwise had no past medical history, allergies, or medications. He was employed as a carpenter in one of the suburbs of a major Texas city prior to his presentation.

On initial examination, he was alert and oriented. Vital signs were stable. His skin was diffusely erythematous and painful to touch consistent with his history of prolonged exposure in the Texas summer weather. His right lower extremity demonstrated a spiral continuous superficial abrasion on the medial and lateral sides of his knee, beginning approximately two to three centimetres below his joint line. The abrasion was superficial and did not communicate with his knee joint or deeper tissues. He had palpable biphasic dorsalis pedis and posterior tibial pulses and his capillary refill was less than two seconds. The posterior portion of his right leg was very firm to touch and was painful with passive extension. His cutaneous sensation was intact below his knee with the exception of the plantar surface of his foot. Initial serum chemistry abnormalities were as follows: sodium 154 mEq/L, BUN 34 mg/dL, creatinine 2.6 mg/dL, AST 349 IU/L, ALT 120 IU/L, creatinine kinase MB Mass 595 ng/ml, creatinine kinase 50,156 IU/L, and white blood cell count 21,700. Urinalysis demonstrated 1+ ketone, 3+ blood, and 1+ leukocyte esterase. He was admitted by the general surgery trauma service directly to the surgical intensive care unit.

The patient required bilevel positive airway pressure on his first hospital day and subsequent intubation for respiratory failure coupled with pneumonia. He received his first haemodialysis on his second hospital day and received a four compartment leg fasciotomy on hospital day number six which demonstrated extensive necrotic muscle, particularly in the anterior and deep posterior compartments of the right leg. Lesser degrees of muscle necrosis were noted in the peroneal compartment and the deep posterior compartment and extensor digitorium longus had to be removed in its entirety. On hospital day number eight, after developing gram positive sepsis, he was taken to the operating room for incision and debridement and partial wound closure with retention

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sutures. On appropriate antibiotic therapy, he stabilized, and on hospital day twenty-two, he was transferred to the physical medicine and rehabilitation service where he progressed to having slight ankle flexion with no dorsiflexion and the ability to walk150 ft. with a front-wheeled walker. At 2 month follow-up, he no longer require dialysis, continue to lack ankle dorsiflexion and continue to require the use of an ankle foot orthotic device.

Discussion

Crush syndrome is a manifestation of extensive muscle damage. It is usually seen during wartime.[3] This syndrome was first reported in civilian victims of the aerial bombardment on London in 1941, who had extremities entrapped by debris.[2,6] During peacetime, the majority of victims are earthquake victims or patients deprived of sensation for long periods of time by drug or alcohol intoxication. This syndrome has also been reported in patients during prolonged general anaesthesia.[1] Patients may also develop this syndrome with an unresolved or untreated compartment syndrome.

Direct trauma is thought to cause cell death through mechanical disruption,[4] followed by myonecrosis and ischemic injury. Oedema due to the increased capillary permeability ensues, which can elevate compartment pressures enough to create compartment tamponade.[16] After the source of compression is released, the necrotic tissue will often reabsorb sodium from the returning blood flow, causing further swelling along with the release of myoglobin, lactic acid, creatinine, creatinine kinase, phosphate, and potassium into the bloodstream.[5] In addition, abundant oxygen is available for recombination into oxygen free radicals such as superoxide and hydroxyl which further damages cell membranes.[8,9] This serves to promote platelet aggregation, microvasculature clotting, anoxia, and continuation of the overall detrimental process.[10,17] Studies have also demonstrated that nitric oxide of macrophage origin accumulates in crush injury musculature.[21] It has also been documentated that nitric oxide originating from macrophages has cytotoxic effects on the tubular system of the kidney.[23]

The literature appears to be somewhat divided on the treatment for a patient who presents with a longstanding crush injury or compartment syndrome as did our patient and unfortunately, there are very few studies of patients with this injury. A retrospective review of 94 patients at the University of Cincinnati concluded that fasciotomy, when performed early, is best. However, when the fasciotomy is performed late, results of limb salvage, compared to early fasciotomy, are similar. For those patients more than thirty-six hours away from their original injury, their rate of limb salvage has been reported at 25%. This study recommended fasciotomy for crush syndrome or late presentation of compartment syndrome regardless of the time of presentation.[15] Of interest, this study failed to find any predictors of post compartment syndrome complications. A mortality rate approaching 55% has been reported when acute renal failure is complicated by infection or other medical conditions.[19] Other reports describe an increase in nosocomial infections as the hospitalization period lengthens, in association with acute renal failure.[13,18]

Early fasciotomy is thought to be helpful because it reduces the load of muscle lysis products transported to the kidney, decreases harmful cytokine production, and decrease the production of oxygen free radicals. However, another school of thought states that delayed fasciotomy for late presentation of crush injury or compartment syndrome simply exposes necrotic muscle as a bacterial culture medium. Infection rates as high as 46% have been reported.[22] A retrospective review of five patients with ischaemic limbs presenting for treatment 35—96 h after injury who underwent fasciotomies at the University of Toronto demonstrated significant mortality and in fact caused that institution to change its policy to one of supportive care only for the acute renal failure and late reconstructive procedures.[7] These authors believe that delayed fasciotomy converts a closed injury to an open injury and that the nerve and muscle damage in the compartment has already occurred. Reis and Michaleson concluded that a closed crush injury should be left closed until definitive demarcation of gangrene has manifested itself. Their reasoning is that the sequelae of infection are much worse than muscle contracture and that modern dialysis is usually sufficient to help the patient survive through the initial acute renal failure.[20] In another series from the Marmara earthquake aftermath, of 40 patients with fasciotomies, 38 (95%) developed wound infections and nine (22%) eventually died from sepsis.[12] Other earthquake experiences have indicated that there is no evidence that fasciotomy improves outcome in patients with crush syndrome,[14] and support the Marmara experience of increased infection rates.[11]

Conclusion

Crush syndrome is a potentially fatal entity. Published surgical opinion is divided on whether early

fasciotomy or supportive care and late reconstructive procedures are best. There are several recent reports of significant morbidity and mortality with either treatment approach. However, based on available evidence from more recent experiences, there appears to be little justification for debridement of muscle compartments that have already become necrotic.

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